

## Carvedilol

### Chemical Properties

CAS No. : 72956-09-3

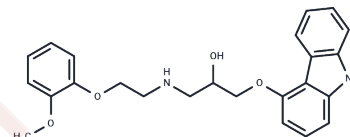
Formula: C<sub>24</sub>H<sub>26</sub>N<sub>2</sub>O<sub>4</sub>

Molecular Weight: 406.47

Store at low temperature

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Actual storage temperature shall be subject to the COA.



### Biological Description

Description	Carvedilol (SKF 105517) Phosphate is the phosphate salt form of carvedilol, a racemic mixture and adrenergic blocking agent with antihypertensive activity and devoid of intrinsic sympathomimetic activity. The S enantiomer of carvedilol nonselectively binds to and blocks beta-adrenergic receptors, thereby exerting negative inotropic and chronotropic effects, and leading to a reduction in cardiac output. In addition, both enantiomers of carvedilol bind to and block alpha 1-adrenergic receptors, thereby causing vasodilation and reducing peripheral vascular resistance.
Targets(IC50)	Antibacterial, Adrenergic Receptor, Autophagy, VEGFR
In vitro	Carvedilol rapidly inhibits Fe(++)-initiated lipid peroxidation, measured as thiobarbituric acid reactive substance (TBARS), in rat brain homogenate with an IC <sub>50</sub> of 8.1 mM. Carvedilol protects against Fe(++)-induced alpha-tocopherol depletion in rat brain homogenate with an IC <sub>50</sub> of 17.6 mM. Carvedilol dose-dependently decreases the intensity of the DMPO-OH signal with an IC <sub>50</sub> of 25 mM. [1] Carvedilol has inverse efficacy for stimulating G(s)-dependent adenylyl cyclase but stimulates phosphorylation of the receptor's cytoplasmic tail on previously documented G protein-coupled receptor kinase sites in beta2 adrenergic receptor (beta2AR)-expressing HEK-293 cells. [2] Carvedilol (0.1-10 mM) produces a concentration-dependent inhibition of the mitogenesis stimulated by platelet-derived growth factor, epidermal growth factor, thrombin, and serum in human cultured pulmonary artery vascular smooth muscle cells, with IC <sub>50</sub> values ranging from 0.3 mM to 2.0 mM. Carvedilol also produces a concentration-dependent inhibition of vascular smooth muscle cell migration induced by platelet-derived growth factor, with an IC <sub>50</sub> value of 3 mM. [3] Carvedilol decreases the extent of cellular vacuolization in cardiac myocytes and prevents the inhibitory effect of doxorubicin on mitochondrial respiration in both heart and liver. Carvedilol also prevents the decrease in mitochondrial Ca(2+) loading capacity and the inhibition of the respiratory complexes of heart mitochondria caused by doxorubicin. [4]

### Solubility Information

Solubility	DMSO: 40.6 mg/mL (99.88 mM), Sonication is recommended. Ethanol: 20.3 mg/mL (49.94 mM), Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
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## A DRUG SCREENING EXPERT

In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (4.92 mM), Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>
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### Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.4602 mL	12.301 mL	24.6021 mL
5 mM	0.492 mL	2.4602 mL	4.9204 mL
10 mM	0.246 mL	1.2301 mL	2.4602 mL
50 mM	0.0492 mL	0.246 mL	0.492 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

### Reference

- Yue TL, et al. J Pharmacol Exp Ther, 1992, 263(1), 92-98.  
Wisler JW, et al. Proc Natl Acad Sci U S A, 2007, 104(42), 16657-16662.  
Ohlstein EH, et al. Proc Natl Acad Sci U S A, 1993, 90(13), 6189-6193.  
Santos DL, et al. Toxicol Appl Pharmacol, 2002, 185(3), 218-227.

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